REVIEW ARTICLE

Management of Acute Type B Aortic Dissection

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A 74-YEAR-OLD MAN PRESENTS TO THE EMERGENCY DEPARTMENT WITH A 1-DAY HISTORY of severe chest and back pain. He has a history of hypertension and has run out of medication. He has no history of similar previous events or a relevant family history. He quit smoking 10 years ago. Pedal pulses are palpable. The results of laboratory tests, including troponin levels, and an electrocardiogram are normal. Computed tomographic (CT) angiography of the chest, abdomen, and pelvis reveals an aortic dissection that extends from the left subclavian artery to the right iliac artery. How should this case be managed?

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CME

Aortic dissection is a catastrophic event that affects approximately 13,000 persons each year in the United States.[1] Most patients with a type B aortic dissection, in which the origin of the aortic entry tear is distal to the left subclavian artery, are initially treated with medications to reduce blood pressure and myocardial contractility. Surgical or endovascular therapy is reserved for complications, such as aortic rupture or end-organ malperfusion. In this review, we describe the current state of the evidence informing clinical practice for the treatment of type B aortic dissection, as well as areas of controversy and future directions.

Acute aortic dissection, intramural hematoma, and penetrating aortic ulcer are three distinct entities encompassed by the clinical diagnosis of an acute aortic syndrome. Aortic dissection starts with a tear in the intima, which might be preceded by medial degeneration and followed by separation and lifting of the intima from the media in an antegrade and retrograde manner. This sequence of events creates two (or occasionally more) channels where the blood can flow outside the true lumen of the aorta, and complications arise if the blood flow into the aortic side branches becomes compromised by the dissection.[2,3]

Acute aortic dissection and intramural hematoma have similar clinical features but different pathophysiological mechanisms. Rupture of the vasa vasorum causes an intramural hematoma, with bleeding into the aortic media. The intramural hematoma may progress to aortic dissection if the ischemic aortic wall ruptures and causes an intimal tear.[4]

EPIDEMIOLOGY

The incidence rate of sporadic aortic dissection is 3 to 4 cases per 100,000 persons per year.[5] The mean age at presentation is 63 years, and men are predominantly affected. There is also a trend toward older age, and among older adults, the incidence among men is similar to that among women.[1,6,7] The risk profile for patients with aortic dissection is related to uncontrolled hypertension.[8] Other contributing factors include connective-tissue diseases, genetic aortopathy, smoking, blunt trauma, and use of illicit drugs, such as cocaine or amphetamines.[9] Delayed diagnosis of acute aortic dissection is not uncommon[10] and is associated with female

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KEY POINTS

MANAGEMENT OF ACUTE TYPE B AORTIC DISSECTION

- Key risk factors for aortic dissection are hypertension and known genetic aortopathy.
- Thoracic endovascular aortic repair (TEVAR) has largely replaced open surgery for patients presenting with complications (rupture or malperfusion).
- Uncomplicated type B aortic dissection is treated medically with blood-pressure control.
- TEVAR or open repair is indicated if complications develop during follow-up.
- Serial imaging and lifelong surveillance are recommended for all patients who have had an aortic dissection.

sex, non-White race, previous cardiac surgery, transfer from another center, and normal blood pressure at presentation.[11,12]

CLASSIFICATION OF ACUTE AORTIC DISSECTION

Acute aortic dissection can be classified according to the location of the intimal tear or according to the affected part of the aorta, irrespective of the site of the tear. The DeBakey classification system was designed to describe the location of the tear, as well as the extent of the affected aorta or the surrounding wall hematoma. According to this classification, a type I aortic dissection originates in the ascending aorta and extends up to at least the aortic arch and usually far distally, beyond the descending thoracic aorta. Type II starts in — and is limited to — the ascending aorta, and type III is limited to the descending and thoracoabdominal aorta. In 1970, Daily, Shumway, and colleagues published the Stanford classification system, which became more widely used.[13] Stanford type A dissections are those involving the ascending aorta; type B are those that do not involve the ascending aorta. Anatomically, the ascending aorta refers to the part of the aorta proximal to, and exclusive of, the innominate artery. Dissections involving the aortic arch but not the ascending aorta are classified and treated as type B. In the classification system of the Society for Vascular Surgery and the Society of Thoracic Surgeons (SVS–STS), the aorta is divided into 12 zones; a type A dissection has an entry tear in zone 0 and extends distally anywhere from zone 1 to zone 12, whereas a type B dissection has an entry tear distal to zone 1 that could extend to zone 12 distally or propagate in a retrograde manner to involve the arch and ascending aorta (Fig. 1).[14]

Up to two thirds of aortic intimal tears originate in the ascending aorta, whereas most of the remaining tears begin close to the left subclavian artery and extend distally. The latter type is termed a type B dissection according to the Stanford and SVS–STS classification systems, most likely because of the development of high shear forces in these locations.[15] Of patients with type B aortic dissections, 60% present without signs of rupture or malperfusion; these dissections are referred to as uncomplicated type B aortic dissections.[16,17]

TREATMENT STRATEGIES AND EVIDENCE

After the diagnosis is made, the initial management of acute type B aortic dissection is aimed at reducing blood pressure and blood-pressure variation over time (Fig. 2).[18] Intravenous beta-blockers, the first-line therapy, are adjusted to maintain systolic blood pressure between 100 and 120 mm Hg and the heart rate between 60 and 80 beats per minute.[19] Opioids, which are usually needed for pain control, may indirectly help control changes in blood pressure. Other beta-blockers or classes of drugs that control blood pressure may be used if monotherapy does not lower the pressure to desired targets.[20] Long-term management includes comprehensive secondary prevention measures, which comprise lifestyle and pharmacologic antihypertensive interventions.[21]

COMPLICATED AORTIC DISSECTION

A type B aortic dissection is considered to be complicated when aortic rupture or end-organ malperfusion is present.[22] Aortic rupture is defined as extravasation of blood outside the confines of the adventitia of the aorta; the blood

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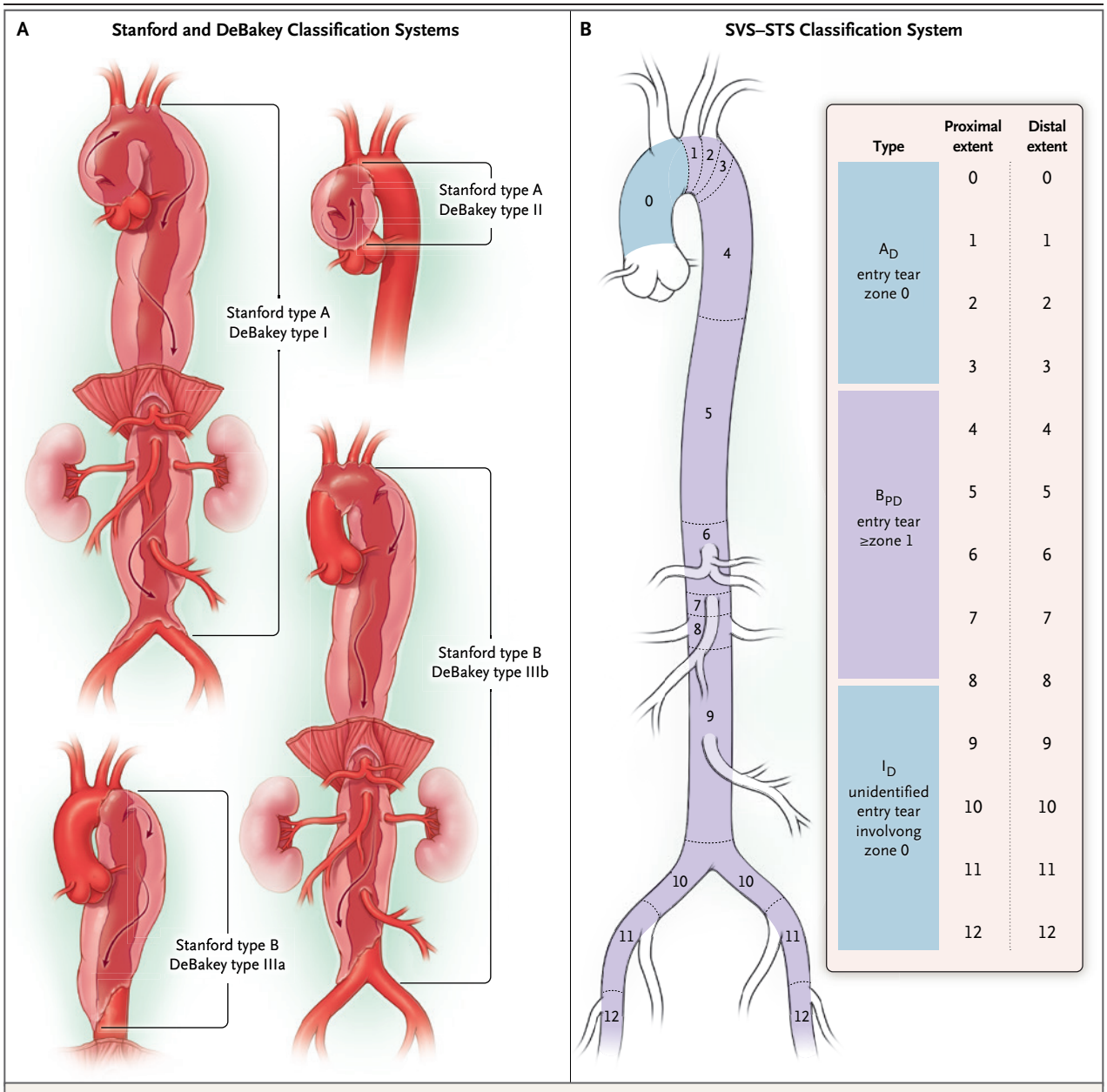

Figure 1. Classification of Aortic Dissection.

Panel A shows the Stanford and DeBakey classification systems. A Stanford type A dissection involves the ascending aorta, which is defined by the proximal border of the innominate artery. All other dissections not involving the ascending aorta are designated as Stanford type B. If the aortic arch is involved but not the ascending aorta, the dissection is classified as type B; rarely, isolated arch dissections occur that are limited to the arch. In the DeBakey system, the dissection is classified as type I if a tear in the ascending aorta propagates distally to the aortic arch and descending aorta, type II if the dissection involves only the ascending aorta, type IIIa if the tear is beyond the ascending aorta and the dissection involves only the descending thoracic aorta, and type IIIb if the tear is beyond the ascending aorta and the dissection involves the entire descending thoracic and thoracoabdominal aorta and the region below the diaphragm. Panel B shows the Society for Vascular Surgery and the Society of Thoracic Surgeons (SVS–STS) classification scheme, which divides the aorta into 12 zones. A type A dissection has an entry tear in zone 0 and extends distally anywhere from zone 1 to zone 12, whereas a type B dissection has an entry tear distal to zone 1 and can extend distally to zone 12 or propagate in a retrograde manner to zone 0. In type I, the entry tear is unidentified and the dissection involves zone 0. D denotes distal extension, and PD proximal or distal extension.

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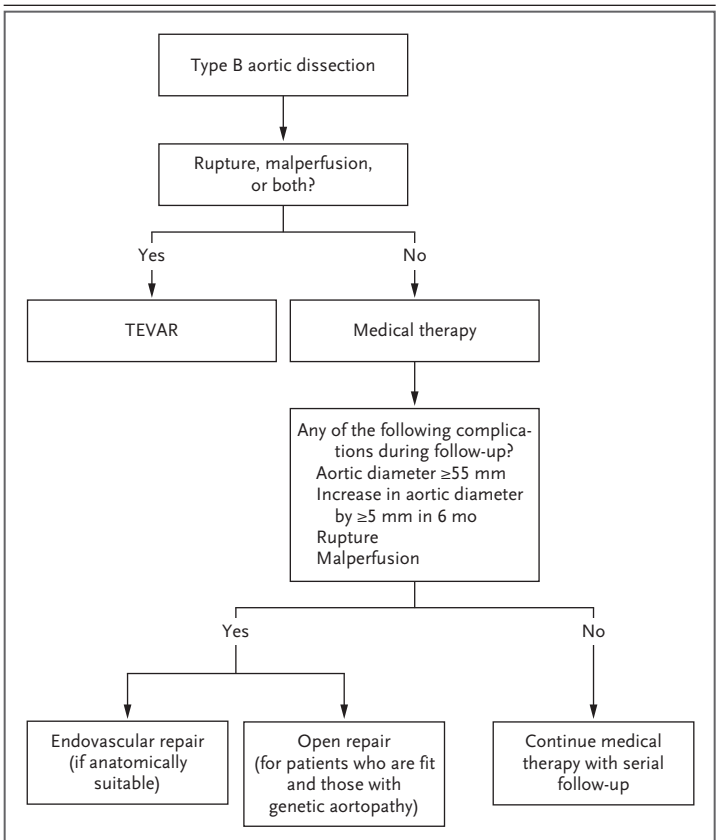

Figure 2. Algorithm for the Management of Type B Aortic Dissection.
Patients with type B aortic dissection who have rupture or malperfusion are generally treated with thoracic endovascular aortic repair (TEVAR). Patients without rupture or malperfusion may be treated with medical therapy alone, but either endovascular or open repair may be warranted if complications develop.

may be released freely or contained by the mediastinal pleura surrounding the aorta. Malperfusion refers to inadequate blood flow to end organs or tissues as a result of branch-vessel obstruction from the aortic dissection, which is detected with imaging.[14] Malperfusion syndrome occurs when malperfusion has progressed to cause tissue or organ ischemia and dysfunction. In addition to characteristic imaging findings, malperfusion syndrome is associated with clinical features and laboratory findings that reflect the compromised vascular territory. Renal malperfusion is the most common type, although nearly all major vascular beds, such as the spinal-cord, mesenteric, and lower-extremity vessels, can be involved.[23]

Malperfusion syndrome is more often due to dynamic obstruction of branch vessels by the dissection (which accounts for 80% of cases) than to static obstruction. The key mechanism involves the mobility of the intimal aortic dissection flap that protrudes into the ostium of a branch vessel and leads to intermittent obstruction of blood flow. This incomplete but repetitive vessel occlusion may produce intermittent symptoms of variable intensity that can at times occur over a period of several days or weeks, which makes diagnosis and management of malperfusion syndrome challenging. Static obstruction of aortic side branches is the result of the false lumen prolapse into the branch vessel, with a resulting branch-vessel thrombosis and overt symptoms that correspond to the compromised vascular territory[24] (Fig. 3).

TIMING AND TYPE OF REPAIR

The 2022 American Heart Association and American College of Cardiology (AHA–ACC) guidelines recommend thoracic endovascular aortic repair (TEVAR) for type B aortic dissection when the anatomy is prone to rupture or other complications.[22] After hemodynamic status has been stabilized, preferably in the intensive care unit, patients with type B aortic dissection who have (or are at risk for) rupture or malperfusion are sent to the operating room or an interventional hybrid suite for emergency open or endovascular repair.

Endovascular repair consists of the placement of a thoracic stent graft that covers the intimal entry tear thought to have triggered the dissection. Often, the proximal end of the thoracic endograft must be placed in zone 2 of the thoracic aorta. In such cases, a carotid–subclavian bypass or branched endograft configuration may be needed to ensure adequate flow into the left subclavian artery in order to reduce the risk of perioperative stroke or arm claudication during follow-up[25] (Fig. 4). Adjunctive stenting of branches of the aorta or distal fenestrations of the aorta septum have also been advocated for persistent end-organ ischemia.[26]

The provisional extension to induce complete attachment (PETTICOAT) technique, which was introduced in 2005 for the treatment of acute, complicated type B aortic dissection, involves the placement of both a stent graft to cover the proximal tear in the aorta and a distal stent to minimize the risk of new tears.[27] In cases of malperfusion, bare stainless-steel stents are usually used to rescue branches into which the dissection flap extends. The technique was tested in the Study of Thoracic Aortic Type B Dissection Using

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Endoluminal Repair (STABLE I and II) in patients with complicated type B aortic dissection. Although the performance goals were met for both the primary effectiveness end point (30-day survival) and the primary safety end point (freedom from major adverse events at 30 days), the long-term incidence of thrombosis of the false lumen in the areas receiving the bare metal stent was unpredictable.[28-30]

To address the inconsistent results of the PETTICOAT technique in inducing aortic remodeling and eliminating false lumen flow, the stent-assisted, balloon-induced intimal disruption and relamination in aortic dissection (STABILISE) technique was introduced.[31] This strategy was adopted initially to accomplish the reattachment of the entire acutely dissected aorta in order to potentially eliminate the need for future interventions.[32,33] However, reports have shown that it might not always be possible to perform the procedure,[34] and it carries an additional risk of rupture when the balloon is inflated inside the dissected aorta.[35] Studies with larger samples and longer follow-up are under way to evaluate the safety and effectiveness of the STABILISE technique.

The open-repair strategy for acute, complicated type B aortic dissection is reserved for patients deemed to be anatomically unsuitable for endovascular repair and those in whom malperfusion persists after TEVAR.[36] In-line or extra-anatomical arterial bypass grafts to occluded visceral or extremity vessels are occasionally placed to restore flow and address end-organ ischemia (Fig. 5). For patients undergoing extensive open replacement or stent-graft coverage of the aorta (the entire thoracic aorta to the celiac trunk), as well as those who have previously undergone aortic surgery or who have occluded collateral vessels, the targets for postoperative systolic blood pressure are often higher than those for patients without these risk factors.[37-39]

UNCOMPLICATED, HIGH-RISK AORTIC DISSECTION

The Food and Drug Administration defines a type B aortic dissection as a tear in the descending aorta that is not associated with vessel rupture or malperfusion. In 2020, the SVS–STS reporting standards introduced the term "high-risk features" of aortic dissections, and this term was subsequently adopted in the guidelines.[14,22] High-risk features include a maximal aortic diameter of more than 4 cm, a false-lumen diameter that exceeds 22 mm,[40] an entry tear of more than 1 cm,[41] an entry tear on the lesser curvature of the aorta, an increase in the total aortic diameter of more than 0.5 cm on serial imaging studies during the index admission, a bloody pleural effusion, imaging-only evidence of malperfusion, refractory hypertension despite administration of more than three classes of antihypertensive medications at maximal recommended or tolerated doses, refractory pain persisting for more than 12 hours despite maximal recommended or tolerated doses of pain medication,[42] and the need for hospital readmission. The data supporting the "high-risk" designation are limited and based on retrospective, small, single-center studies with incomplete follow-up.[40,42,43]

EVIDENCE FOR ENDOVASCULAR TREATMENT OF UNCOMPLICATED AORTIC DISSECTION

Evidence from randomized trials investigating the use of endovascular treatment for uncomplicated type B aortic dissection comes from the Investigation of Stent Grafts in Aortic Dissection (INSTEAD) and the Acute Dissection Stent Grafting or Best Medical Treatment (ADSORB) trial.[44,45] INSTEAD randomly assigned 140 patients with late subacute or chronic uncomplicated type B aortic dissection to undergo medical therapy plus TEVAR or medical therapy alone. Cumulative survival at 2 years was similar in the two groups (P=0.15 by log-rank test).

The long-term results of INSTEAD were evaluated in INSTEAD-XL.[46] Survival estimates diverged after 2 years, and at 5 years, the mean (±SD) aortic dissection–specific mortality was 6.9±3.0% with TEVAR, as compared with 19.3±4.8% with medical therapy alone (hazard ratio, 0.35; 95% confidence interval [CI], 0.13 to 0.98; P=0.04). At 5 years, disease progression had occurred in 27% of patients in the TEVAR group as compared with 46% in the medical-therapy group (hazard ratio, 0.55; 95% CI, 0.32 to 0.98; P=0.04). Landmark analysis at 2 years showed that TEVAR was associated with lower all-cause mortality than medical therapy (0% vs. 16.9%; P=0.0003), as well as with lower aortic dissection–specific mortality (0% vs. 16.9%; P=0.0005) and a lower percentage of patients with disease progression (4.1% vs. 28.1%; P=0.004). At 5 years, complete thrombosis of the false lumen at the stented segment was observed in 90.6% of patients who underwent

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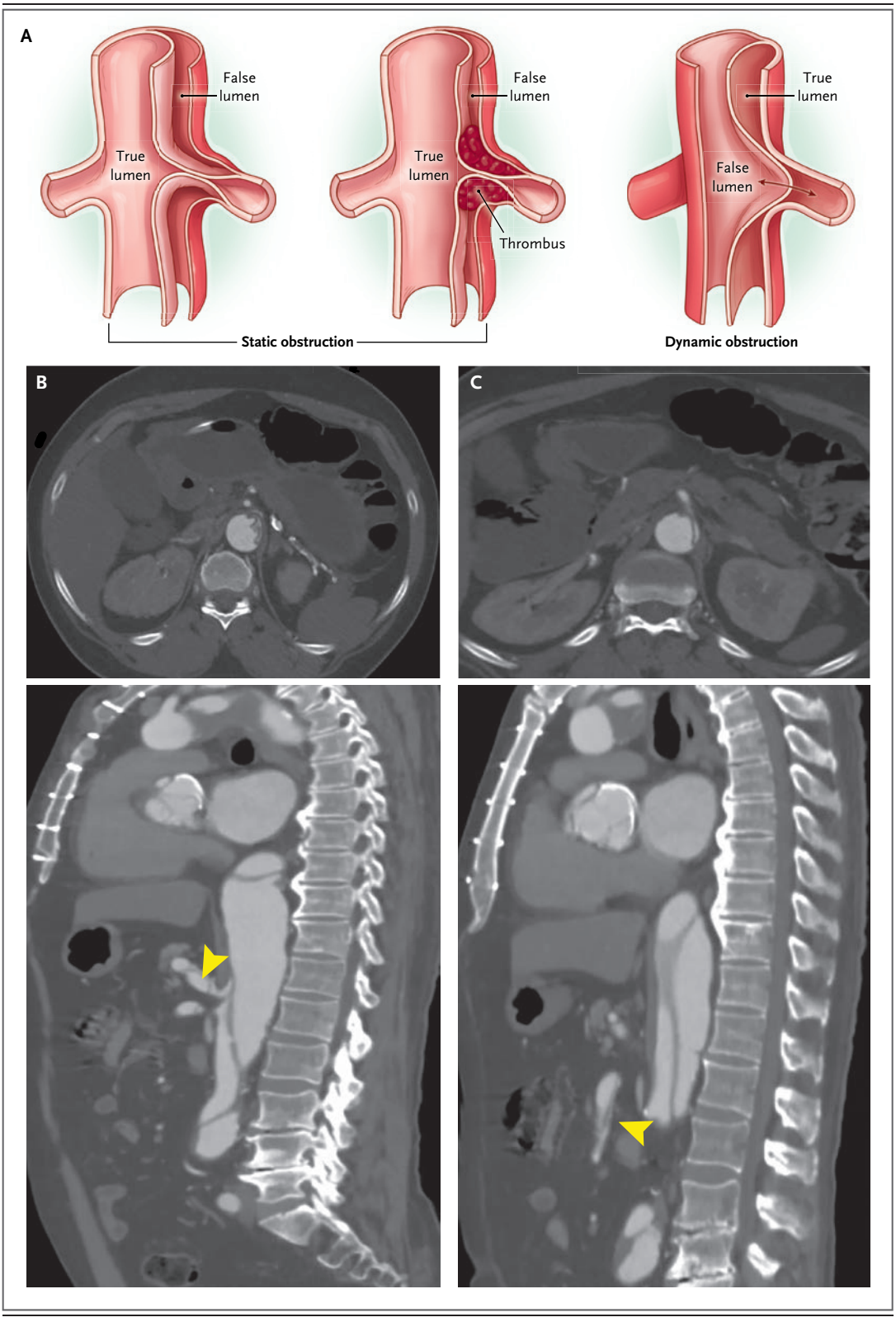

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Figure 3 (facing page). Mechanisms of End-Organ Ischemia.

Panel A shows the mechanisms by which an aortic dissection may lead to end-organ ischemia, including static obstruction, in which the side branch is occluded completely by the dissection flap, with or without thrombus. Dynamic branch obstruction occurs when the dissection flap intermittently occludes the side branch. The axial and sagittal computed tomography (CT) images in Panel B (top and bottom, respectively) show malperfusion of the celiac artery caused by an aortic dissection. The arrowhead indicates the false lumen that has extended into the celiac artery. The axial and sagittal CT images in Panel C (top and bottom, respectively) show malperfusion of the superior mesenteric artery caused by an aortic dissection. The arrowhead points to the compressed true lumen that has extended into the superior mesenteric artery.

TEVAR, as compared with 22.0% of patients treated with medical therapy alone (P<0.0001).

In the ADSORB trial, which involved patients with acute type B aortic dissection (with onset <14 days before enrollment), 31 patients were randomly assigned to receive medical therapy alone and 30 patients to undergo TEVAR plus medical therapy. At 1 year of follow-up, remodeling of the aorta was seen in 57% of the patients in the TEVAR group, as compared with 3% of those in the medical-therapy group (P<0.001).[47] For TEVAR to have a meaningful role in the management of uncomplicated type B aortic dissection, it must be accomplished with minimal risk of procedure-related complications such as stroke, paraplegia, aortic rupture, retrograde aortic dissection, and the development of new distal stent-

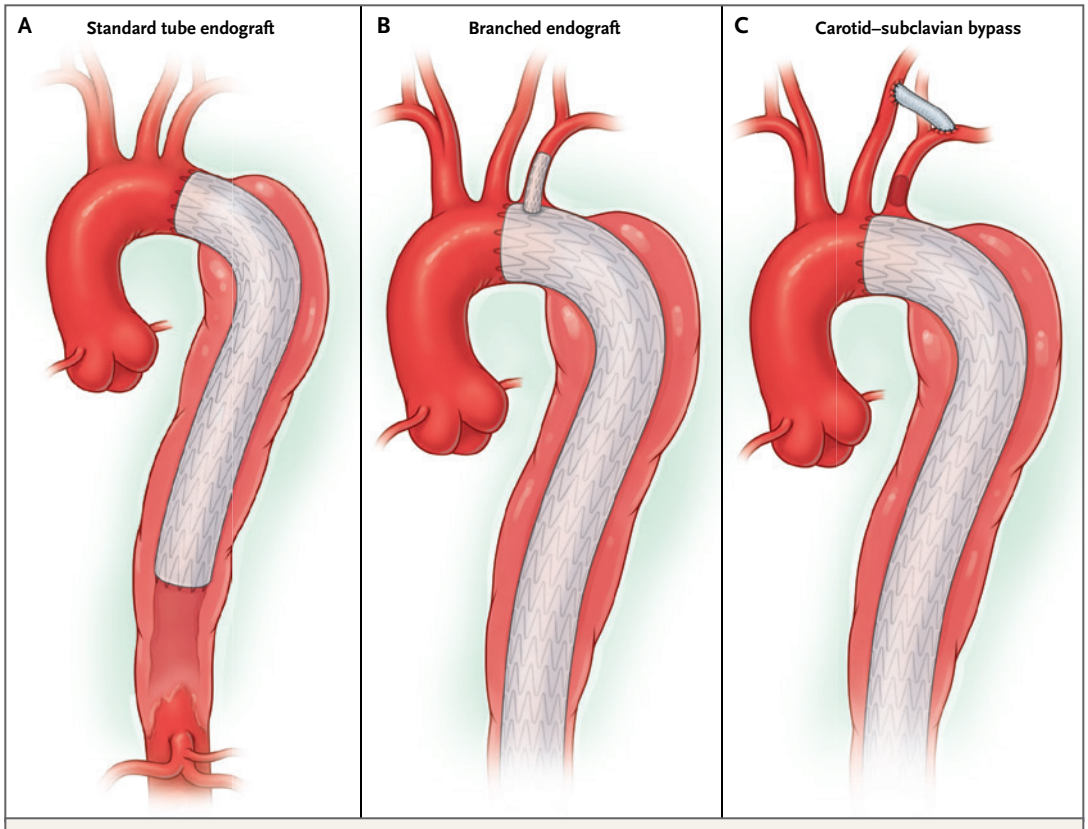

Figure 4. Examples of TEVAR.

Panel A shows an example of standard zone 3 TEVAR to cover a proximal entry tear. Panel B shows zone 2 TEVAR with a thoracic branched endograft to ensure flow into the left subclavian artery. Panel C shows zone 2 TEVAR, with a carotid–subclavian arterial bypass to ensure flow into the subclavian artery and an endovascular plug to occlude the origin of the left subclavian artery to prevent future type II endoleak. Zones refer to SVS–STS classification.

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induced entry tears. It also must mitigate the risk of future aneurysmal degeneration.[48]

LONG-TERM IMAGING PROTOCOL

Lifelong radiologic surveillance is warranted to detect any progression of type B aortic dissection, to assess the durability of open or endovascular repair, and to detect any signs of aneurysmal degeneration of the false lumen, which occurs in 25 to 40% of cases, as well as remodeling of the aorta with expansion of the true lumen and obliteration of the false lumen.[49] After discharge, all patients with type B aortic dissection are encouraged to follow a guideline-directed clinical and imaging follow-up protocol, with repeat imaging at 1, 6, and 12 months after the initial diagnosis and then yearly thereafter. During follow-up, intervention is required if a rupture develops and is recommended if the total aortic diameter increases by 5 mm in 6 months or by 10 mm in 1 year or if it reaches 55 mm.[22]

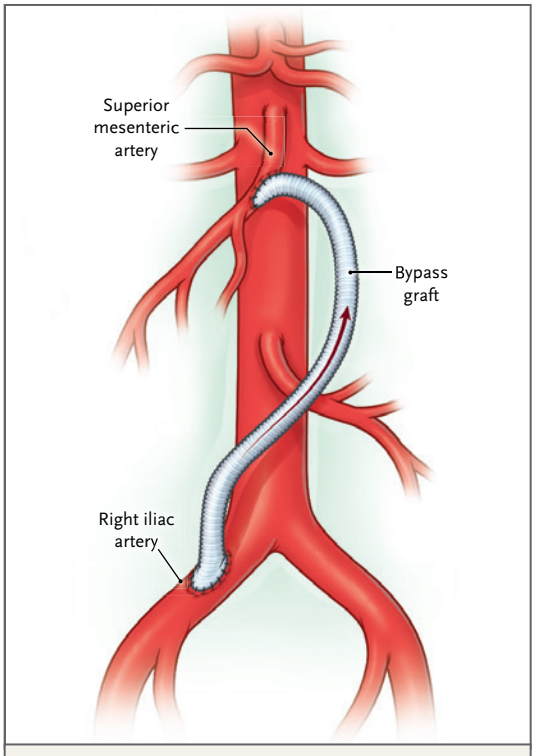

Figure 5. Right Iliac–Superior Mesenteric Arterial Bypass for Malperfusion.

Retrograde iliac–superior mesenteric arterial bypass may be used when TEVAR or branch stenting does not resolve malperfusion. The arrow indicates the retrograde flow.

AREAS OF UNCERTAINTY

ROLE OF EARLY TEVAR FOR UNCOMPLICATED, HIGH-RISK AORTIC DISSECTION

The role of TEVAR in patients with high-risk features remains speculative and is determined by provider preference, because this strategy has not been evaluated with the use of a robust prospective study design. Given the adoption of the high-risk features in several sets of guidelines, however, there is an urgent need to enroll patients in randomized, controlled trials in order to generate high-quality data and scientifically validate these features.

INTERVENTION FOR UNCOMPLICATED TYPE B AORTIC DISSECTION

In addition to the presence of high-risk features, other factors have a role in determining the management approach and have led to variability in clinical decision making with regard to the indications and justification for an operative intervention, as well as the timing and extent of the procedure.[50,51] Key considerations include the ability of the patient to adhere to follow-up serial imaging and an antihypertensive regimen, the long-term effects of the use of iodinated contrast material needed for follow-up imaging, and institutional expertise.

TIMING AND EXTENT OF TEVAR FOR UNCOMPLICATED AORTIC DISSECTION

The most effective timing of TEVAR in patients with uncomplicated type B aortic dissection is unclear. However, the general consensus is that very early intervention (<48 hours after admission) is best avoided to minimize the chance of complications, particularly the development of a type A retrograde aortic dissection.[52,53]

The extent of thoracic aortic coverage is based on several considerations. Since there is no malperfusion associated with uncomplicated type B aortic dissection, most interventionalists opt to cover the entry tear and extend distally at various distances, depending on the complexity and extent of the dissection.[54] The choice of the length of the aorta to be covered with a stent graft reflects an effort to balance the intended goal of sealing the dissection flap against aortic-wall remodeling and concern about the possible development of spinal-cord ischemia.[55]

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GENETICALLY TRIGGERED THORACIC AORTIC DISEASE

Genetically triggered thoracic aortic disease is a term that encompasses several syndromic and nonsyndromic conditions, including Marfan's syndrome, the Loeys–Dietz syndrome, vascular Ehlers–Danlos syndrome, and nonsyndromic heritable thoracic aortic disease.[19,56] Traditionally, TEVAR was considered to be contraindicated in patients with genetically triggered thoracic aortic disease because of arterial-wall fragility, the risk of creating a retrograde type A dissection during or after the procedure, and the high rates of short- and long-term failure. For these reasons, uncomplicated type B aortic dissection in patients with genetically triggered thoracic aortic disease is typically treated conservatively with medical therapy. Complicated type B aortic dissection poses distinct treatment challenges, with open repair favored over endovascular repair. However, TEVAR has been used as a temporizing lifesaving measure in anticipation of a more definitive open repair in the future.[57]

PREGNANCY

Aortic dissection is rare during pregnancy, and the diagnosis may be delayed because of a myriad of symptoms, as well as concern about maternal and fetal radiation exposure in the case of a first-trimester pregnancy.[58-60] A multidisciplinary management strategy should follow institutional protocols.[22] In general, uncomplicated type B aortic dissection during pregnancy is treated medically with labetalol.[61] Treatment of complicated type B aortic dissection during pregnancy is the same as treatment in the nonpregnant state, with emergency intervention.[62,63]

GUIDELINES

The most recent guidelines for the management of thoracic aortic dissection have been published by the AHA–ACC[22] and by the American Association for Thoracic Surgery and the STS.[64] Although both sets of guidelines assign a class 1 recommendation to medical therapy, up-front TEVAR in selected patients with uncomplicated type B aortic dissection has a class 2b recommendation.

Given the lack of data from adequately powered randomized trials with respect to the outcomes and cost-effectiveness of up-front TEVAR in patients with uncomplicated type B aortic dissection, including those with high-risk features, the leadership of both the SVS and the European Society for Vascular Surgery have endorsed an effort to prioritize research to inform the use of TEVAR and have led the efforts that culminated in funding of the Improving Outcomes in Vascular Disease — Aortic Dissection (IMPROVE-AD) trial in the United States; the Scandinavian Trial of Uncomplicated Aortic Dissection Therapy (SUNDAY) in Scandinavia, Australia, New Zealand, and selected sites in Europe; and the Early Aortic Repair in Patients Needing Endovascular/Open Surgery for Type B Aortic Dissection (EARNEST) trial in the United Kingdom.

CONCLUSIONS

The patient described in the vignette in the introduction has several risk factors for aortic dissection, including male sex and a history of smoking and hypertension, and he presents with severe chest and back pain. The CT angiogram is diagnostic of a type B aortic dissection. He has palpable distal pulses and no clinical or laboratory evidence of malperfusion, which indicates the presence of uncomplicated type B aortic dissection. Current practice includes admission to the intensive care unit for pain management and strict control of blood pressure and heart rate with antihypertensive therapy, along with close clinical and laboratory follow-up in order to allow for early detection of signs of malperfusion or rupture. The development of abdominal or leg pain or worsening chest pain would warrant immediate CT angiography of the chest, abdomen, and pelvis to assess the patient for malperfusion or aortic rupture. If such an acute event developed, TEVAR with coverage of the entry tear would be appropriate. Otherwise, lifelong surveillance with clinical examination and serial imaging to detect aneurysmal degeneration is recommended, with treatment, when appropriate.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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